WEST Search History

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DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L1	phoshatonin$	1	L1
DB=USPT; PLUR=YES; OP=OR			
L2	L1	0	L2
DB=PGPB; PLUR=YES; OP=OR			
L3	L1	1	L3

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=> s phosphatonin?/bi,ab 24 PHOSPHATONIN?/BI 21 PHOSPHATONIN?/AB
L1 24 PHOSPHATONIN?/BI,AB

=> s l1 not 2003/py 402621 2003/PY
L2 22 L1 NOT 2003/PY

=> s l2 not 2002/py 1064337 2002/PY

L3 18 L2 NOT 2002/PY

=> d l3 1-18 bib ab

L3 ANSWER 1 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2001:895738 CAPLUS
DN 137:60903
TI Hypophosphatemia and rickets/osteomalacia
AU Tanaka, Hiroyuki
CS Department of Pediatrics, Okayama University Graduate School of Medicine and Dentistry, Japan
SO Clinical Calcium (2001), 11(10), 1282-1289 CODEN: CLCCEJ; ISSN: 0917-5857
PB Iyaku Janarusha
DT Journal; General Review
LA Japanese
AB A review. It is important to know the pathophysiol. of the inherited hypophosphatemic disorders for the research on the relationship between hypo phosphatemia and

rickets/osteomalacia. And the research on the pathogenesis of the X-linked hypophosphatemic vitamin D resistant rickets/osteomalacia has played central roles in this field. After the identification of PHEX gene for the responsible gene of the disorder, major interest of the research has shifted to the identification of phosphatonin , the natural substrate of PHEX and the novel phosphate-regulating hormone. Recent discovery of FGF23 for the responsible gene of oncogenic osteomalacia and autosomal dominant hypophosphatemic rickets may lead our knowledge on the phosphate regulation system to the new stage.

L3 ANSWER 2 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2001:696078 CAPLUS
DN 136:245768
TI Evidence for a PTH-independent humoral mechanism in post-transplant hypophosphatemia and phosphaturia
AU Green, Jacob; Debby, Hilla; Lederer, Eleanor; Levi, Moshe; Zajicek, Hubert K.; Bick, Tova
CS Department of Nephrology, Rambam Medical Center, B. Rappaport Faculty of Medicine, Technion, Israel Institute of Technology, Haifa, Israel
SO Kidney International (2001), 60(3), 1182-1196 CODEN: KDYIA5; ISSN: 0085-2538
PB Blackwell Science, Inc.
DT Journal
LA English
AB Patients undergoing successful kidney transplantation often manifest overt hypophosphatemia assocd. with exaggerated phosphaturia during the early post-transplant period (2 wk to 3 mo). The mechanism for this phenomenon was not fully elucidated. We tested the hypothesis that a circulating serum factor [non-parathyroid hormone (non-PTH)], which operates during chronic renal failure (CRF) to maintain phosphate (Pi) homeostasis, can increase fractional excretion of Pi (FEPO4) in normal functioning kidney grafts during the early post-transplant period, thereby causing phosphaturia and hypophosphatemia. 5 Groups of patients were studied: control subjects (group 1, N = 16), "early" (2 wk to 1 mo) post-transplant patients (group 2, N = 22), "late" (9 to 12 mo) post-transplant patients (group 3, N = 14), patients with advanced CRF (glomerular filtration rate = 30 to 40 mL/min; group 4, N = 8), and patients who suffered from end-stage renal failure and were treated by chronic hemodialysis (group 5, N = 14). Group 2 manifested significant hypophosphatemia and phosphaturia when compared with groups 1 and 3 (Pi = 0.9 .+-. 0.003 mg/dL, FEPO4 = 68 .+-. 5%, P < 0.0005 vs. groups 1 and 3). Sera were taken from each of the 5 subject groups and applied to the proximal tubular opossum kidney (OK) cells. The activity of Na/Pi-type 4 (i.e., OK-specific type II transporter) was evaluated by measuring Na+-dependent 32Pi flux. The expression of Na/Pi type II mRNA and the abundance of Na/Pi protein were detd. by Northern and Western blot assays, resp. When compared with sera from groups 1 and 3, 10% sera taken from groups 2, 4, and 5 (incubated overnight with OK cells) inhibited 32Pi flux by 25 to 30% (P < 0.0003). Both Na/Pi mRNA and the expression of Na/Pi protein were markedly augmented under the same conditions (P < 0.05 groups 2, 4, and 5 vs. groups 1 and 3). Time-course anal. revealed that the up-regulation of Na/Pi protein by sera from groups 2, 4, and 5 was obsd. as early as 4 h of incubation, whereas augmented abundance of Na/Pi mRNA was only seen after 8 h of incubation. The addn. of PTH (1-34) to sera from groups 2, 4, and 5 abolished the augmented expression of NaPi protein. We labeled OK cell surface membrane proteins with N-hydroxysuccinimide bound to biotin (NHS-SS-biotin). Biotinylated transporters incubated with the different sera were pptd. by strepavidin and identified by Western blot anal. Cells incubated in sera from group 2 showed increased membrane bound transporter when compared with control sera, whereas the intracellular pool of the transporter was comparable between the 2 groups. A non-PTH circulating serum factor (possibly phosphatonin) that increases FEPO4 during CRF is also responsible for phosphaturia and hypophosphatemia in the early period following successful kidney transplantation. The putative factor inactivates Na/Pi activity along with inhibition of the transporter trafficking from the cell membrane into the cytosol.
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2001:643574 CAPLUS
DN 135:193521
TI Hypophosphatemic rickets/osteomalacia
AU Fukumoto, Seiji
CS Dep. Lab. Med., Univ. Tokyo Branch Hosp., Japan
SO Horumon to Rinsho (2001), 49(8), 743-749 CODEN: HORIAE; ISSN: 0045-7167
PB Igaku no Sekaisha
DT Journal; General Review
LA Japanese
AB A review with 32 refs., on the clin. symptoms, pathogenesis, diagnosis, and treatment of hypophosphatemic rickets/osteomalacia (HR). Topics discussed include: disorders in phosphate reuptake by renal proximal tubule and vitamin D metab., mutations in PHEX gene in X-linked HR, role of phosphatonin in the hypophosphatemia, mutations in FGF23 gene in autosomal dominant HR, overprodn. of FGF23 in tumor-induced rickets/osteomalacia, and pathogenesis of hereditary HR with hypercalciuria.

L3 ANSWER 4 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2001:580342 CAPLUS
DN 135:286097
TI Molecular aspects of phosphate homeostasis in mammals
AU Beck, L.; Silve, C.
CS INSERM U 426 et Institut federatif de recherche .mchlt. Cellules Epitheliales .mchgt., Faculte de medecine Xavier Bichat, Paris, Fr.
SO Nephrologie (2001), 22(4), 149-159 CODEN: NEPHDY; ISSN: 0250-4960
PB Medecine et Hygiene
DT Journal; General Review
LA French
AB A review with refs. Renal phosphate reabsorption, the major determinant of phosphate homeostasis, is primarily dependent on dietary phosphate content and multiple hormonal factors. Over the last few years, the identification of sodium-dependent phosphate transporters in kidney, intestine and bone, as well as new insights into the mol. mechanisms involved in several hereditary hypophosphatemias, allow to set up novel phosphate reabsorption regulatory pathways. This review describes mol. players involved in these mechanisms, summarizes phosphate transport data in kidney, intestine and bone, and describes recent findings concerning the three most common hereditary hypophosphatemias.
RE.CNT 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 18 CAPLUS COPYRIGHT 2003 ACS

AN 2001:439564 CAPLUS
DN 135:193641
TI FGF-23 Inhibits Renal Tubular Phosphate Transport and Is a PHEX Substrate
AU Bowe, Ann E.; Finnegan, Richard; Jan de Beur, Suzanne M.; Cho, Justin; Levine, Michael A.; Kumar, Rajiv; Schiavi, Susan C.
CS Applied Genomics, Genzyme, Framingham, MA, 01701-9322, USA
SO Biochemical and Biophysical Research Communications (2001), 284(4), 977-981 CODEN: BBRCA9; ISSN: 0006-291X
PB Academic Press
DT Journal
LA English
AB Oncogenic osteomalacia (OOM), X-linked hypophosphatemia (XLH), and autosomal dominant hypophosphatemic rickets (ADHR) are phenotypically similar disorders characterized by hypophosphatemia, decreased renal phosphate reabsorption, normal or low serum calcitriol concns., normal serum concns. of calcium and parathyroid hormone, and defective skeletal mineralization. XLH results from mutations in the PHEX gene, encoding a membrane-bound endopeptidase, whereas ADHR is assocd. with mutations of the gene encoding FGF-23. Recent evidence that FGF-23 is expressed in mesenchymal tumors assocd. with OOM suggests that FGF-23 is responsible for the phosphaturic activity previously termed " phosphatonin ". Here we show that both wild-type FGF-23 and the ADHR mutant, FGF-23 (R179Q), inhibit phosphate uptake in renal epithelial cells. We further show that the endopeptidase, PHEX, degrades native FGF-23 but not the mutant form. Our results suggest that FGF-23 is involved in the pathogenesis of these three hypophosphatemic disorders and directly link PHEX and FGF-23 within the same biochem. pathway. (c) 2001 Academic Press.
RE.CNT 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2001:401694 CAPLUS
DN 135:135857
TI FGF23, hypophosphatemia, and rickets: has phosphatonin been found?
AU Strewler, Gordon J.
CS Department of Medicine, Veterans Affairs Boston Healthcare System and Harvard Medical School, Boston, MA, 02132, USA
SO Proceedings of the National Academy of Sciences of the United States of America (2001), 98(11), 5945-5946 CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
AB A polemic with T. Shimada et al. (ibid. 2001, 98, 6500) is given, concerning identification of FGF23 (member of the fibroblast growth factor family) as the humoral factor that is secreted by tumors to cause tumor-induced osteomalacia. Pathogenesis of renal phosphate wasting and the discovery that a protease mutation and a cleavage site mutation cause the same disease are discussed. FGF23 is supposed not to be the final phosphatonin but to stimulate secretion of a final phosphate-regulating factor. Although the other 22 FGFs share only 4 known receptors, FGF23 is supposed to have a different receptor because cleavage of its COOH terminus inactivates it.
RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2001:126413 CAPLUS
DN 134:293719
TI The autosomal dominant hypophosphatemic rickets (ADHR) gene is a secreted polypeptide overexpressed by tumors that cause phosphate wasting
AU White, Kenneth E.; Jonsson, Kenneth B.; Carn, Gwenaelle; Hampson, Geeta; Spector, Tim D.; Mannstadt, Michael; Lorenz-Depiereux, Bettina; Miyauchi, Akimitsu; Yang, In Myung; Ljunggren, Osten; Meitinger, Thomas; Strom, Tim M.; Juppner, Harald; Econs, Michael J.
CS Dept. of Medicine, Indiana University School of Medicine, Indianapolis, IN, 46202, USA
SO Journal of Clinical Endocrinology and Metabolism (2001), 86(2), 497-500 CODEN: JCEMAZ; ISSN: 0021-972X
PB Endocrine Society
DT Journal
LA English
AB The gene mutated in autosomal dominant hypophosphatemic rickets (ADHR), a phosphate wasting disorder, has been identified as FGF-23, a protein that shares sequence homol. with fibroblast growth factors (FGFs). Patients with ADHR display many of the clin. and lab. characteristics that are obsd. in patients with oncogenic hypophosphatemic osteomalacia (OHO), a disorder thought to arise by the secretion of a phosphate wasting factor from different mesenchymal tumors. In the present studies, we therefore investigated whether FGF-23 is a secreted factor and whether it is abundantly expressed in OHO tumors. After transient transfection of OK-E, COS-7, and HEK293 cells with the plasmid encoding full-length FGF-23, all three cell lines efficiently secreted two protein species into the medium that were approx. 32 and 12 kDa upon SDS-PAGE and subsequent Western blot anal. using an affinity-purified polyclonal antibody to FGF-23. Furthermore, Northern blot anal. using total RNA from five different OHO tumors revealed extremely high levels of FGF-23 mRNA, and Western blot anal. of exts. from a sixth tumor detected the 32 kDa FGF-23 protein species. In summary, FGF-23, the gene mutated in ADHR, is a secreted protein and its mRNA is abundantly expressed by several different OHO tumors. Our findings indicate that FGF-23 may be a candidate phosphate wasting factor, previously designated " phosphatonin ".
RE.CNT 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 8 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2001:121278 CAPLUS
DN 135:74747
TI Rickets: It's not just vitamin D deficiency
AU Hochberg, Ze'ev
CS Department of Pediatrics, Rambam Medical Center, Haifa, 31096, Israel
SO Current Opinion in Endocrinology & Diabetes (2001), 8(1), 23-28 CODEN: CENDES; ISSN: 1068-3097
PB Lippincott Williams & Wilkins
DT Journal; General Review
LA English
AB A review with 41 refs. Malnutrition of vitamin D and Ca is still the most common cause of rickets and esp. in individuals with dark complexions. The capacity of the 2 renal enzymes in vitamin D metab. is rather low. Only approx. 10% of 25-

hydroxyvitamin D is 24-hydroxylated and 0.3% is 1 .alpha.-hydroxylated. The activities of these 2 enzymes are reciprocally controlled by 1,25-dihydroxyvitamin D itself, and vitamin A antagonizes the action of vitamin D. Mutations in the 25-hydroxyvitamin D-1.alpha.-hydroxylase gene cause hereditary vitamin D-dependent rickets type I (VDDRI). The domains that are essential for the enzyme activity were identified and are rather selective. X-linked hypophosphatemic rickets is caused by mutations in the PHEX gene and inactivity of the membrane-bound endopeptidase, resulting in impairment of both Na-dependent P-cotransporter and vitamin D metab. Likewise, tumor-induced osteomalacia is related to overprodn. by a mesenchymal tumor of the putative phosphatonin with remission after resection of the tumor. These disease mechanisms lead to a rational algorithmic approach to diagnosis of rickets.
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 9 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2000:605031 CAPLUS
DN 134:145256
TI Tumor-induced osteomalacia and the regulation of phosphate homeostasis
AU Kumar, R.
CS Departments of Medicine, Biochemistry, and Molecular Biology, Mayo Clinic and Foundation, Rochester, MN, USA
SO Bone (New York) (2000), 27(3), 333-338 CODEN: BONEDL; ISSN: 8756-3282
PB Elsevier Science Inc.
DT Journal; General Review
LA English
AB A review with 72 refs. Tumor-induced osteomalacia (TIO) is a rare and unique syndrome characterized by hypophosphatemia, excessive urinary phosphate excretion, reduced 1,25-dihydroxyvitamin D concns., and osteomalacia. Removal of the tumor is assocd. with a cure of the lesion. Several labs. have now shown that conditioned medium derived from cultures of such tumors contain a small, heat-sensitive substance (" phosphatonin ") of <25,000 Da that specifically inhibits sodium-dependent phosphate transport in cultured renal proximal tubular epithelia. This substance does not increase cAMP formation in tubular epithelial cells and does not increase cAMP excretion in urine. A substance with similar properties is present in the circulation of patients on hemodialysis. A syndrome with a remarkably similar biochem. phenotype, namely, X-linked hypophosphatemic rickets (XLH), also has a circulating factor with properties similar, if not identical, to those of the tumor-derived factor, " phosphatonin .". The mol. defect in XLH has been shown to be due to a mutant endopeptidase, PHEX, whose substrate might be " phosphatonin .". Hypophosphatemia and other biochem. abnormalities in TIO are due to excessive prodn. of " phosphatonin " with normal PHEX function, whereas the biochem. abnormalities in XLH are caused by a mutant PHEX enzyme that fails to process " phosphatonin .".
RE.CNT 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 10 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2000:297675 CAPLUS
DN 133:206023
TI PHEX gene and hypophosphatemia
AU Drezner, Marc K.
CS Departments of Medicine and Cellular Biology, Duke University Medical Center, Durham, NC, USA
SO Kidney International (2000), 57(1), 9-18 CODEN: KDYIA5; ISSN: 0085-2538
PB Blackwell Science, Inc.
DT Journal; General Review
LA English
AB A review, with 54 refs. X-linked hypophosphatemia (XLH) and tumor-induced osteomalacia (TIO) are diseases that have in common abnormal proximal renal tubular function resulting in increased renal clearance of inorg. phosphorus and hypophosphatemia. The recent discovery of the PHEX gene has provided new insights to these disorders. In this regard, identification of the PHEX gene product as a membrane-bound endopeptidase suggests that the pathophysiol. cascade underlying XLH likely involves inactivation mutations of the gene causing a failure to clear an active hormone, phosphatonin , from the circulation. The presence of this hormone through unknown mechanisms decreases the sodium-dependent phosphate cotransporter in the kidney, resulting in impaired phosphate transport. In contrast, TIO likely evolves secondary to tumor overprodn. of the putative phosphatonin , which exerts physiol. function despite efforts to counteract the resultant hypophosphatemia with overprodn. of PHEX transcripts that are insufficient to accommodate the enhanced substrate load. These potential pathophysiol. mechanisms for XLH and TIO provide valuable inroads to understanding phosphate homeostasis, as well as vitamin D metab., bone mineralization, and calcium metab.
RE.CNT 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 11 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2000:229403 CAPLUS
DN 133:162237
TI Mutations in PHEX gene and X-linked hypophosphatemic rickets
AU Miyamoto, Kenichi
CS School of Medicine, Tokushima University, Japan
SO Igaku no Ayumi (2000), 192(6), 678-681 CODEN: IGAYAY; ISSN: 0039-2359
PB Ishiyaku Shuppan
DT Journal; General Review
LA Japanese
AB A review with 8 refs. on regulation of phosphate level in the blood, structure and mutations of the gene PHEX protein, phosphatonin , and mutations in the PHEX gene and X-linked hypophosphatemic rickets.

L3 ANSWER 12 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 2000:47186 CAPLUS
DN 132:332921
TI Coordinated maturational regulation of PHEX and renal phosphate transport inhibitory activity: evidence for the pathophysiological role of PHEX in X-linked hypophosphatemia
AU Nesbitt, Teresa; Fujiwara, Ikuma; Thomas, Ronald; Xiao, Zhou-Sheng; Quarles, L. Darryl; Drezner, Marc K.
CS Departments of Medicine, Pathology, and Cell Biology, Duke University Medical Center, Durham, NC, USA
SO Journal of Bone and Mineral Research (1999), 14(12), 2027-2035 CODEN: JBMREJ; ISSN: 0884-0431
PB Blackwell Science, Inc.
DT Journal
LA English
AB The mechanism by which inactivating mutations of PHEX (phosphate- regulating gene with homologies to

endopeptidases on the X chromosome) cause X-linked hypophosphatemia remains unknown. However, recent reports suggest errant PHEX activity in osteoblasts may fail to inactivate a phosphaturic factor produced by these cells. To test this possibility, the authors examd. coordinated maturational expression of PHEX and prodn. of phosphate transport inhibitory activity in osteoblasts from normal and hyp-mice. The authors assessed the inhibitory activity in conditioned medium by examg. the effects on opossum kidney cell phosphate transport and osteoblast PHEX expression by reverse transcriptase-polymerase chain reaction during a 17-day maturational period. Inhibitory activity increased as a function of osteoblast maturational stage, with no activity after 3 days and persistent activity by 6 days of culture. More significantly, equal phosphate transport inhibitory activity in conditioned medium from normal and hyp-mouse osteoblasts (control 1.90, normal 1.48, hyp 1.45 nmol/mg of protein/min) was obsd. at 6 days. However, by 10 days hyp-mouse osteoblasts exhibited greater inhibitory activity than controls, and by 17 days the difference in phosphate transport inhibition maximized (control 2.08, normal 1.88, hyp 1.58 nmol/mg of protein/min). Concurrently, the authors obsd. absent PHEX expression in normal osteoblasts after 3 days, limited prodn. at 6 days, and significant prodn. by day 10 of culture, while hyp-mouse osteoblasts exhibited limited PHEX activity secondary to an inactivating mutation. The data suggest that the presence of inactivating PHEX mutations results in the enhanced renal phosphate transport inhibitory activity exhibited by hyp-mouse osteoblasts.
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 13 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 1999:460532 CAPLUS
DN 131:128268
TI Oncogenic osteomalacia
AU Miyauchi, Akimitsu
CS Dep. Intern. Med., Natl. Sunat. Hyogo Chuo Hosp., Japan
SO Nippon Naika Gakkai Zasshi (1999), 88(7), 1287-1290 CODEN: NNGAAS; ISSN: 0021-5384
PB Nippon Naika Gakkai
DT Journal; General Review
LA Japanese
AB A review with 11 refs., on differential diagnosis, clin. symptoms, pathogenesis, and treatment of tumor-induced vitamin D resistant osteomalacia (oncogenic osteomalacia). Pathogenic role of humoral phosphaturic factor, phosphatonin , produced by tumor cells is also discussed.

L3 ANSWER 14 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 1999:233136 CAPLUS
DN 131:30336
TI Sodium-dependent phosphate co-transporters
AU Takeda, Eiji; Taketani, Yutaka; Morita, Kyoko; Miyamoto, Ken-Ichi
CS Department of Clinical Nutrition, School of Medicine, University of Tokushima, Tokushima, 770-8503, Japan
SO International Journal of Biochemistry & Cell Biology (1999), 31(3/4), 377-387 CODEN: IJBBFU; ISSN: 1357-2725
PB Elsevier Science Ltd.
DT Journal; General Review
LA English
AB A review with 19 refs. The renal proximal tubular resorption of inorg. phosphate (Pi) mediated by sodium-dependent phosphate (Na+/Pi) co-transporters plays a crit. role in the maintenance of Pi homeostasis. Two nonhomologous Na+/Pi co-transporters (type I and type II) have been identified in the renal cortex of various species. The role of the type I co-transporter in Pi regulation remains to be clarified. Type II co-transporters play a major role in the regulation of renal Pi resorption by dietary Pi and parathyroid hormone, which regulate the rapid endocytosis/exocytosis of the transporters. Type III Na+/Pi co-transporters, which are expressed in a wide variety of tissues and are regulated by changes in the Pi concn., have recently been described. The presence of a novel Pi-regulating hormone called " phosphatonin " has been postulated in studies of the mechanisms of X-linked hypophosphatemic rickets and oncogenic osteomalacia. The regulation of phosphatonin and Na+/Pi co-transporters may provide novel pharmacol. approaches to the treatment of these diseases.
RE.CNT 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 1999:191449 CAPLUS
DN 130:221170
TI Oncogenic osteomalacia and phosphatonin
AU Fukumoto, Seiji
CS Branch Hosp., Univ. Tokyo, Japan
SO Horumon to Rinsho (1999), 47(3), 253-257 CODEN: HORIAE; ISSN: 0045-7167
PB Igaku no Sekaisha
DT Journal; General Review
LA Japanese
AB A review with 14 refs., on the diagnosis, treatment, and etiol. of oncogenic osteomalacia with hypophosphatemia. Pathogenic role of phosphatonin , a phosphaturic factor, produced by tumor cells is also discussed.

L3 ANSWER 16 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 1999:40138 CAPLUS
DN 130:79438
TI Pathology of tumor-induced osteomalacia and rickets
AU Ishida, Tsuyoshi; Kashima, Kenji; Machinami, Rikuo
CS Fac. Med., Univ. Tokyo, Tokyo, 113-8655, Japan
SO Byori to Rinsho (1999), 17(1), 23-27 CODEN: BYRIEM; ISSN: 0287-3745
PB Bunkodo
DT Journal; General Review
LA Japanese
AB A review with 21 refs. about pathol. of tumor-induced osteomalacia and rickets. Their histol. characteristics and variety and a relationship between so-called " phosphatonin " as a causing substances, and phosphate regulating gene with homologies to endopeptidases located on the X chromosome (PEX) are discussed.

L3 ANSWER 17 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 1997:705418 CAPLUS
DN 127:344192
TI New concepts concerning the regulation of renal phosphate excretion
AU Kumar, Rajiv
CS Dept. of Internal Medicine, Division of Nephrology, Mayo Clinic, Rochester, MN, 55905, USA
SO News in Physiological Sciences (1997), 12(Oct.), 211-214 CODEN: NEPSEY; ISSN: 0886-1714
PB American Physiological Society
DT Journal; General Review
LA English

AB A review with 15 refs. Some tumors elaborate a phosphaturic factor, " phosphatonin ," that is unlike known peptide or sterol hormones. The same, or a similar, factor may exist in hypophosphatemic mice and humans with X-linked hypophosphatemic rickets. Such a factor may play a role in the control of phosphate homeostasis in normal physiol. states.

L3 ANSWER 18 OF 18 CAPLUS COPYRIGHT 2003 ACS
AN 1997:167022 CAPLUS
DN 126:247077
TI Phosphatonin -a new phosphaturetic hormone? (lessons from tumor-induced osteomalacia and X-linked hypophosphatemia)
AU Kumar, R.
CS Division of Nephrology, Mayo Clinic and Foundation, Rochester, MN, 55905, USA
SO Nephrology, Dialysis, Transplantation (1997), 12(1), 11-13
CODEN: NDTREA; ISSN: 0931-0509
PB Oxford University Press
DT Journal
LA English
AB There is good evidence that a new factor , phosphatonin , may be responsible for the alteration in phosphate transport seen in various diseases such as tumor-induced osteomalacia, X-linked hypophosphatemia, and hypophosphatemia in the Hyp mouse and the gyroscopic mouse. Phosphatonin may be abnormally low in patients with tumoral calcinosis, an unusual disease assocd. with elevated serum phosphorus and 1,25-dihydroxyvitamin D concns., and ectopic deposition of calcium and phosphorus. Phosphatonin may be appropriately elevated in patients with renal failure who have phosphate retention.

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FULL ESTIMATED COST 51.19 51.40

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE TOTAL ENTRY SESSION
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